2.5.M

MNR MEDICAL COLLEGE

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Name of the Examination : _____ Date: 27/10/23

Subject : Biochemistry Paper : _____ Part : _____

Signature of the Candidate : _____ Roll No : 13

Maximum Marks	10	10	4	4	4	4	4	2	2	2	2	2	50
Q. No.	1	2	3	4	5	6	7	8	9	10	11	12	Total
Marks Secured													

Grand Total :

Marks Secured in Words

50/100

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1) The mineral deficiency seen here is calcium.
Sources of calcium are cereals, pulses, vitamin D.

Absorption of calcium:-

Calcium is actively absorbed in the part duodenum. Calcium is actively transported into blood.

Factors maintaining homeostasis of calcium:-

Para Thyroid Hormone, calciterol, calcitonin maintains the homeostasis of calcium in the blood

calciterol:-

The vitmamin D (1,25 dihydroxy calciferol) helps in maintaining calcium levels It depends on the hormone PTH regulates the calcium levels.

calcitonin:- calcitonin reduces the calcium levels by acting on the bone causing bone deformation. Reducing the bone mass osetophonic called osteoclast cells.

PTH plays very important role in the body. calcium homeostasis

There are two types of cells called osteoclast and osteoblast. osteoblast helps in bone formation

deficiency reduces the bone mass. This cause bone demineralisation. (osteoporosis) reduction in the bone mass.

Calcium is required high quantity for the Pregnancy and lactating women.

Adult normal ca level is 200- 300 mg/dl and for lactating and pregnancy women is 600- 800 mg/dl. Due to some factors calcium level varies in blood. If calcium level increased in blood it is hypercalcimia if it is decreased it is hypocalcimia. In Hypocalcimia due to production of more calcium it causes excretion of more calcium in urine.

Due to the disorders of calcium this female is suffering with pain in hip joint, wrist joints cramps in calf muscles and easy fatigueability

Calcium is important for its main functions as.

- Transport of enzymes
- muscle contraction
- Integrity of cell membrane
- Release of some hormone
- formation of calmodulin complex

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- Transport of substances across membrane
- Activation function
- Erythrocytes for fragility of RBC cells
- membrane integrity
- Transport of nerve impulse
- Blood coagulation

Ca calmodulin formation helps in transport of hormones. Osteoblastic is decrease bone mass is due to decrease blood calcium levels.

Tetany is disorder of muscle weakness due to the accumulation of Ca in the muscle.

Hypocalcemia causes nausea, vomiting, VIT B_6, D supplements are given for the correction hypocalcemia.

PTH, calcitonin, calciferol play important role in maintain role of calcium homeostasis in the blood. Osteoblasts helps in bone formation. Osteoclasts cause bone demineralization / deformation.

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β- oxidation of Palmitic Acid:-

β- oxidation is the process of oxidation of fatty acids.

β- oxidation occurs in the site of cytosol and mitochondria. Transport of acetyl CO-A from cytosol to mitochondria occurs by Carnitine cycle.

β- oxidation of palmitic acid operated cycle of β- oxidation for 6 times

palmitic acid + 6ATP + 6NADP + 6TP + 6H2O →
6CO2 + 6ADP + 6H+ 6NAD

∴ 6ATP — 6 × 2.5 = 90 ATP
FAD — 1.5 = 6 × 1.5 = 70

and there is loss of 2 ATP.

162 - 2 = 106 ATP.

so, there are 106 ATP are produced each palmitic Acid.

ATP produced from the palmitic cycle is 106

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cytosol

Pyruvate

CoASH / TPP

PDH complex.

Acetyl CoA

This Acetyl CoA produced in cytosol is transported to mitochondria by carnitine transport system.

Carnitine transport system :-

① Carnitine phospho transferase - I

② Carnitine phospho transferase - II

Acetyl CoA

|

Carnitine Acyl con.

Carnitine phospho transferase I

outermembrane

Carnitine acyl con

|

Acetyl con

Carnitine phospho transfer II

Inner membrane.

Acetyl con is not permeable to mitochondrial membrane. to make it more through the inner membrane. acetyl con participate in carnitine transport chain for permeable to the mitochondrial membrane

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Acetyl CoA

| ketoisomerase

β- keto - Acyl CoA

| β- hydroxy Acyl CoA synthase

β- hydroxy Acyl CoA

| Acyl synthase

Acyl CoA Acetyl CoA → Krebs Cycle → CO_2.

Acetyl CoA transpoles from cytosol to mitochondria by carnitine transport system. -Carnitine phospho transfuse I converts acetyl CO-A carnitine Acetyl the transports from outer membrane to intermembrane carnitine phospho transfuse II transports the Carnitine Acetate to Acetyl CoA transpose to Inner membrane & mitochondria Acetyl CoA cannot cross the membrane it is impermeable by carnitine transport system it enters as Acetyl CoA into mitochondria

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Acetyl CoA enters into mitochondria by carnitine transport system. Acetyl CoA converts to the β-keto Acyl CoA by intramolecular, By the Enzyme β-hydroxy Acyl CoA synthase β-keto Acyl CoA converts to β-hydroxy Acyl CoA. By synthase β-hydroxy Acyl CoA converts into Acyl CoA and Acetyl CoA Acetyl CoA enters the krebs cycle This cycle is operated for 6 times for precursor as palmitic acid.

There are 108 ATP generated by the palmitic Acid pathway.

2 ATP are used during this cycle

108 - 2 = 106

Therefore 106 ATP are generated through the palmitic cycle

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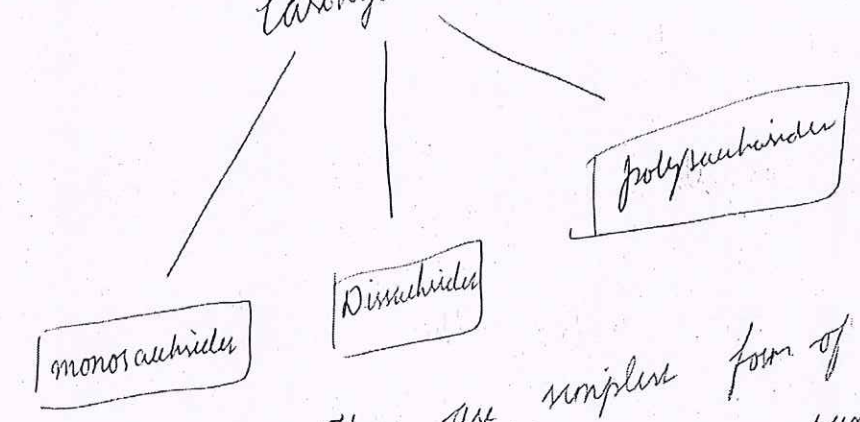
Carbohydrates

Carbohydrates are poly hydroxy aldehydes / ketones or substances which on hydrolysis give aldehydes / ketones. Carbohydrates are also called saccharides. Based on the number of carbon atoms carbohydrates divided into the types



Monosaccharides:- These are simplest form of the carbohydrate. They are made of one type of unit.

Example:- Glucose, fructose, Lactose :-

Glucose is present in the energy formation

fructose is present in the fruits and honey.

Lactose is a milk protein.

Disaccharides:- They are made by two monosaccharides they are complex than the monosaccharides

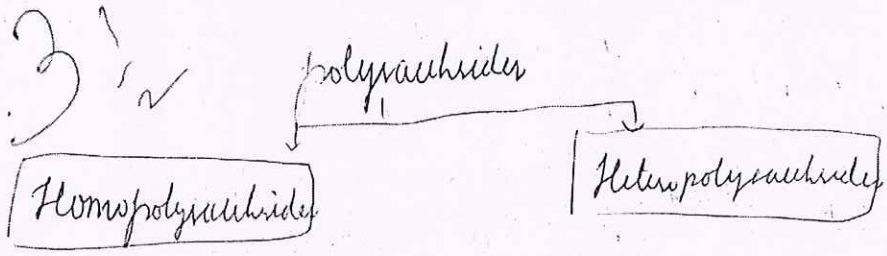
Example - Galactose, Sucrose, Mannose

polysaccharides are more complex than the disaccharides

Glucose + Lactose — Galactose

Glucose + Glucose — Mannose.

Fructose + Fructose — Sucrose.



polysaccharides with same type of the repeating units are homopolysaccharides and they consist different types of monosaccharide are heteropolysaccharide

Homopolysaccharide example is cellulose made of only same repeating unit of glucose.

Heteropolysaccharide example is Starch it contains two structures that are amylose and Amylopectin

Amylose is not branched α 1,4 linkages it consists of the α1,4 linkages

amylopectin is branched structure it consists of both α1,4 and α1,6 linkages

Gluconeogenesis :-

Synthesis of glucose from the non-carbohydrate compounds is known as gluconeogenesis.

Synthesis of glucose from the lactose is the cori cycle. Lactose + ADP + Pi → glucose + ATP.

Lactose form the pyruvate that form the glucose.

Hormonal regulation :-

Insulin and dephosphorylation increase gluconeogenesis

Glucagon and phosphorylation decrease the process of gluconeogenesis

Gluconeogenesis synthesis from the non-carbohydrate source like lipoproteins the glucose is synthesis this process is reverse of the glycolysis exception of few steps which are irreversible.

Pyruvate

Phiolase

Glucose

Gluconeogenesis is the process of synthesis of glucose from the non carbohydrate precursors. Ex:- 9/ glucose is synthesised from the Lactate the process of gluconeogenesis is Cori cycle. Insulin has negative role in Gluconeogenesis. phosphorylation decreases gluconeogenesis. dephosphorylation increases the process of gluconeogenesis.

---

7) Liver showed enlarged hepatocytes with Glycogen accumulation in the hepatocytes.

Glycosemia is excretion of the glycogen in the blood. 2 1/2

Glycosemia :- Accumulation of glycogen in the urine. The production of glycogen is same but its absorption is decreased.

Glycosuria :- Formation of oxalate stones in the kidney is the glycosemic symptom of type B is supplied here.

Gluconeogenesis is the process of synthesis of glucose from the non-carbohydrate precursor cells. Like - 9% glucose is synthesised from the Lactose the process of gluconeogenesis is Cori cycle. Insulin has negative role in gluconeogenesis. phosphorylation decreases gluconeogenesis. dephosphorylation increases the process of gluconeogenesis.

---

7). Liver showed enlarged hepatocytes with Glycogen accumulation in the hepatocytes Glycosuria is excretion of the glycogen in the blood. 2/2

Glycosuria :- Accumulation of glycogen in the urine. The production of glycogen is same but its absorption is decreased.

Glycosuria :- Formation of oxalate stones in the kidney is the glycolysis supplies to it. Which is supplied here.

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Glycogen is synthesised by the UDP- glucose.
Glucose - 6 - phosphate converts to glucose - 1 - phosphate
to UDP glucose by the glycogenin primer
glycogen process. Extension and branching forms
Glycogen. Glycogen breakdown is glycogenolysis
It occurs in liver and muscle. In liver it
forms Glucose. but in muscle due to absence of
Glucose - 6 phosphatase Glucose - 6 - phosphate is
End product in the muscle. glycogen is
converted to the limit dextrin it is then
breakdown glycogen - 6 - phosphate - to the glucose
6 - phosphate. The Enzyme glucose - 6 - phosphate
converts Glucose - 6 - phosphate to glucose in liver
while the enzyme is absent in the
muscle. Glucose 6 - phosphate is the
product in the muscle

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Prostaglandins

Prostaglandins are leukocytes, autacoids
autacoids these are known as prostaglandins
prostaglandins and their compounds regulate
many body functions. thromboxanes leukotrienes
are the components of prostaglandins.

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11. Anion Gap

The total cation concentration — Total anion concentration is Anion gap

80% of the Anions consits of the HCO3 and Cl- remaining 20% are Anion vacancy by the other anionic substance

— Normal value of Anion gap is

15 MEq/litre

Cation concentration = Anion conc + Anionic vacancy.

cation conc - Anion conc = Anion gap.

Anion conc and cation conc is generally equal this is used for maintaining the Acid - base balance.

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12. Methyl groups are the source of the one carbon compounds. Folyen helps in the one carbon metabolism. Methyl group carbon compounds that contains 1 carbon is the structure

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13. Excess carbohydrates converted to the fats synthesis of the fatty acids. Excess are converted to fat and stored as adipose tissue and utilizes whenever it is needed. carbohydrates converted to fatty and stored in the adipose tissue. the mechanism for regulation is fatty acid synthesis

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Cholesterol Synthesis

Acetyl CoA → HMG CoA

↓

Mevalonate

↓

Isopentenyl unit

↓

Squalene

↓

Lanosterol

↓

Cholesterol (37°C)

Cholesterol synthesis in five steps process

1) Regulation of HMG-CoA
2) Synthesis of mevalonate
3) Squalene
4) Lanosterol
5) Synthesis of cholesterol

Acetyl CoA

↓ HMG CoA synthase

HMG CoA

↓ HMG CoA dehydrogenase

mevalonate

↓ mevalonate phosphatase

mevalonate phosphatase

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Mevalonate pyro phosphate

↓ CO_2 pyro phosphatase

3. phospho - 3 pyrophosphomevalonate

↓

Dimethyl allyl pyrophosphate

↓ farnesyl synthase

farnesyl.

↓

Squalene

↓

Lanosterol

↓

Cholesterol

Cholesterol synthesis is increased by the phosphorylation. Cholesterol synthesis is decreased by the dephosphorylation. Hormones regulation glucagon increase whereas insulin decrease the cholesterol synthesis

15) Bile salts and Bile pigments are produced by the liver. Bile pigments are bilirubin and biliverdin. Bile salts are synthesized by the liver.

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16) Galactosemia

Accumulation of the glycogen in the blood is called as Galactosemia. Galactose concentration increases in the blood more than normal limit. Due to disease is the galactose absorption into cells its concentration is increased in the blood. Galactosemia is the increase of conc of Galactose in the blood.

17) During starvation glucose is not present to meet the energy requirements of the body. ketone body synthesis overcome to energy requirements ketone bodies synthesis is better than fatty acids synthesis. ketone bodies require ATP and transport easily and freely

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Acetoacetyl CoA

β-hydroxy-β-methyl glutaryl CoA

β-hydroxy butyrate dehydrogenase

Acetone

β-Hydroxy butyrate

Ketonuria can overcome by the ketone body synthesis

18) Accumulation of the lipids in the tissues / cells is known as storage disease. Gaucher disease is known when excess lipids are present in the body.

19) Electrophoresis :-

Transport of cations and anions to their respective electrodes is Electrophoresis. Anions are negatively charged particles move to the positive electrode Anode, cations are positively charged particles move towards cathode negatively charged

Electrophoresis

Paper Electrophoresis

Gel Electrophoresis

(1) They separate the plasma protein into five bands albumin, α_1, α_2, β & γ

(2) The is more precise stands into 10 bands,

→ Immune Electrophoresis

— Depends on the Antigen an Antibody reaction

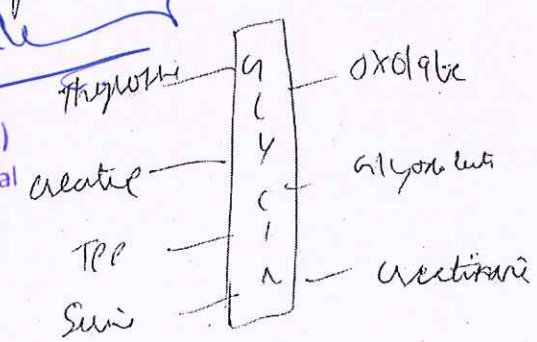
Separation

---

20) Biomolecule derived from glycine

Serine, Thyroxine, oxolate, glyoxolate are produced from glycine. TPP, Collagen, creatine and to hippuric acid. They participate in conjugated reactions with cholic acid. Chenodeoxycholic acid. They are used in the structure of nucleotides

Serine, Thyroxine, oxolate, fumarate, glyoxalate, creatine



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43 1/2

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Essay Questions :-

2) β Oxidation :-

→ Oxidation of fatty acids mainly occurs by β oxidation
→ β oxidation is the oxidation of fatty acid on the β carbon
→ β Oxidation steps are :-
* Activation of fatty acids
* Transportation of fatty acids to mitochondria
* β oxidation proper in mitochondria

Activation of fatty acids :-

→ fatty acids are first activated to acyl coA
→ This step is a two step mechanism
→ This step requires coenzyme A, ATP, Mg^{+}
→ The fatty acid first binds with ATP and forms acyadenyl ate and then it binds with coA to form acyl coA
→ ATP is utilized during this step, two highly energy phosph ate are liberated
→ inorganic phosphate converts this PPi to phosphate
→ The immediate conversion of PPi to phosphate makes this step irreversible

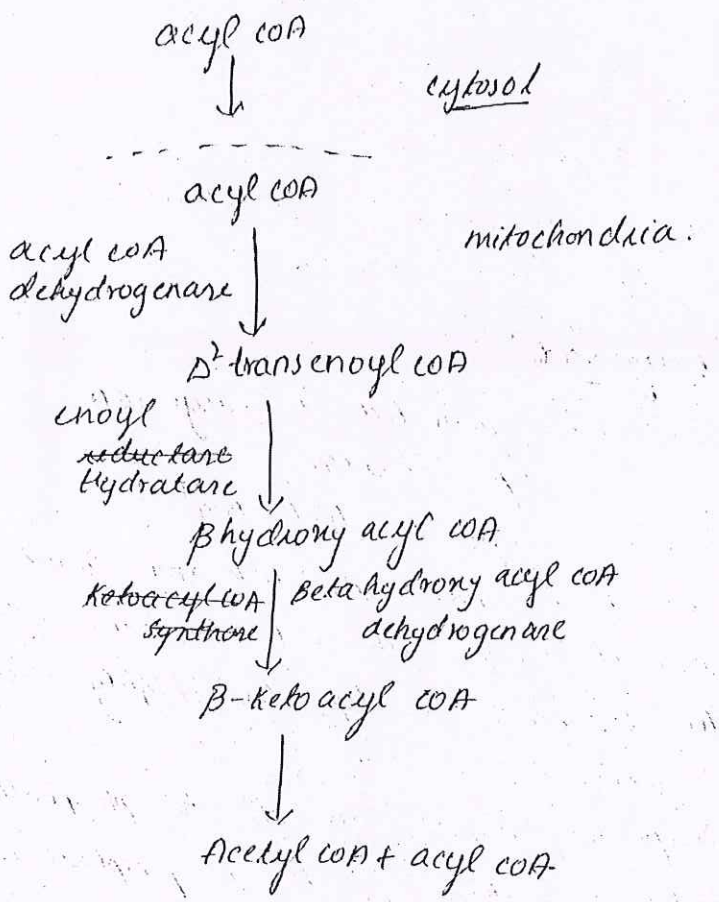
Transport to mitochondria :-

→ fatty are acids cannot pass through inner mitochondrial membrane
→ inner mitochondrial membrane is impermeable to fatty acids so, carnitine system helps in the transport of fatty acids
→ This occurs through 4 steps
* fatty acids will get attached to carnitine through carnitine acyl transferase – I (which is present on the outer mitochondrial membrane)
* Carnitine fatty acid complex will pass through mitochon drial membrane
* On the mitochondrial membrane, the carnitine-acyl transfer ase II seperates the carnitine-acely coA complex

into carnitine and acetyl CoA acyl CoA

→ The carnitine again crosses back the mitochondrial membrane to again bind with acetyl acyl CoA

B Oxidation proper in mitochondria :-



Oxidation :-

→ acyl CoA oxidizes to form Δ^2 transenoyl CoA catalyzed by acyl CoA dehydrogenase

→ there is a double bond formation.

Hydrogenation :-

→ Δ^2 transacyl CoA is converted Betahydroxy acyl CoA catalyzed by enoyl Hydratase

→ double bond is Hydrated

Oxidation :-

→ β hydroxy acyl CoA is converted to β-keto acyl CoA catalyzed by Beta hydroxy acyl CoA dehydrogenase

Cleavage :-

→ Beta ketoacyl CoA is cleaved to form acetyl CoA and acyl CoA

→ Ultimately this reaction is for the production of

Acetyl CoA

Summary:-

C_n Acyl CoA + FAD + NAD + CoASH + H_2O ⟶ $C_{(n-2)}$ Acyl CoA + Acetyl CoA + $FADH_2$ + NADH

→ This reaction continues until fatty acids are completely oxidised.

β Oxidation of palmitic acid :-

Palmitic acid + 7 NAD + 7 FAD + 7 CoASH + $7H_2O$ ⟶ 8 Acetyl CoA + 7 NADH + 7 $FADH_2$ + $14H^+$

→ Palmitic acid undergoes β oxidation for 7 times till the production of 8 molecules of Acetyl CoA

→ This acetyl CoA then enters the TCA cycle for further oxidation.

Odd chain fatty acid Oxidations:-

→ β oxidation of odd chain fatty acids will be same as that of even chain fatty acids except the last and final step

→ In the last and final step 3 carbon molecule is left behind instead of 2 carbon molecule

→ Propionyl CoA is left behind

Propionyl CoA —(Propionyl CoA carboxylase, ATP → ADP)→ Methyl malonyl CoA (D-form) —(Methyl malonyl CoA racemase)→ Methyl malonyl CoA (L-form) —(Methyl malonyl CoA mutase, Vit. B_{12})→ Succinyl CoA

→ Propionyl CoA is carbonylated to methyl malonyl CoA in the D form catalyzed by propionyl CoA carboxylase

→ Methyl malonyl CoA is changed to methyl malonyl CoA (in L form) by Methyl malonyl CoA racemase,

→ Only methyl malonyl coA (L-form) can enter the metabolic pathway

→ Methyl malonyl coA (L form) is then converted to succinyl coA by Methyl malonyl coA mutase, this reaction requires vitamin B_{12}

---

1) Osteopenia is due to the deficiency of mineral calcium

→ Calcium:-

→ calcium is the most abundant mineral present in the body.

→ body contains about 1-1.5 kg of calcium in which 99% is mostly present in bone and teeth & and that 1% is present in other tissues which has wide functions.

functions of calcium:-

→ Development of bone and teeth:-
calcium and phosphorus are required for the formation of hydroxyapatite and bone is the mineralized form of connective tissue

→ Bone in the dynamic state is the storage site for calcium.

→ Blood coagulation:-
→ Many factors in the cascade of Blood coagulation is dependent on calcium.

→ Nerve transmission:-
→ Nerve impulses transmission requires the help of calcium as neurotransmitters.

→ Muscle contraction:-
→ calcium interacts with troponic c and triggers the muscle contraction
→ It also increases the activity of ATPase which increases the interaction between actin and myosin.

→ Activation of enzymes:-
→ calcium directly activates enzymes. for eg:- lipase
succinyl coA dehydrogenase

→ Calmodulin mediated calcium activation :-

-> calmodulin is the ca-binding regulatory protein
-> calmodulin-ca complex activates enzymes

→ Membrane integrity :-

-> ca interfaces with membrane surface and helps in the transportation of water, electrolytes and ions

→ action on heart :-

-> calcium acts on myocardium of heart and prolongs the systole

Calcium absorption :-

→ calcium absorption occurs in the duodenum
→ many factors affect the calcium absorption.

factors that favour calcium absorption :-

→ vitamin D :-

vitamin D / calcitriol it stimulates the synthesis of calcium binding proteins in the intestinal cells

* It increases the uptake of calcium.

→ Parathyroid hormone :-

Parathyroid hormone stimulates the synthesis of calcitriol and inturn increases the calcium uptake

→ Acidity :-

- Acidic nature favours calcium uptake

→ Lactose :-

Lactose cause ca-binding proteins in the intestinal cell to uptake calcium.

→ Lysin and arginine also favours calcium uptake

factors that inhibit calcium uptake :-

→ Phytates and oxalates form insoluble masses with calcium and inhibits the uptake of calcium.

→ increase in dietary intake of phosphorus :- decreases the calcium intake as phosphorus form calcium phosphate which is an insoluble mass.

→ Alkaline nature :-

Alkaline environments inhibits the uptake of calcium.

→ free fatty acids forms insoluble calcium soaps which inhibits the calcium uptake

→ dietary fibres also inhibit the calcium uptake

Sources :-

Best sources :- milk and milk products.

Good sources :- Green leafy vegetables, pulses, cereals, cabbage

Requirements :-

adult man :- 800 mg/day

Women in lactation and pregnancy :- 1-1.5 g/day

children :- 1-1.2 g/day

infantile :- 300-500 mg/day

Plasma calcium level :-

→ Plasma contains calcium more as red blood cells do not contain / contain in less quantities of Ca (9-11 mg/dl)

→ About half of it is in ionized form which is the most active form.

→ Plasma calcium level is mainly maintained by calcitriol, parathyroid harmones and calcitonin.

Calcitriol :-

→ Active form of vitamin D is a harmone called calcitriol or 1,2 dihydroxy cholecalciferol

→ calcitriol stimulates the synthesis of ca-binding regulatory proteins in the intestinal cells.

→ this increases the Blood calcium level

→ calcitriol further increases the uptake of calcium by osteoblasts and causes calcification and remodeling

Parathyroid harmones :-

→ Parathyroid harmones is produced by a part of parathyroid gland which has close relationship

with thyroid gland
-> It consists of 84 amino acids.
-> Originally parathyroid gland is synthesized as prepro parathyroid gland.
-> later it is degraded and forms pro PTH and then to active PTH
-> The secretion and formation is regulated by low concentration of Calcium
-> The production of PTH is a negative regulation feed back mechanism in relation to concentration of calcium

Mechanism of action :-

-> Parathyroid hormone first attaches to the specific receptors on the cell surface
-> This activates the adenyl cyclase which intern activates cyclic AMP
-> The causes increase in calcium which causes of phosphorylation of proteins, hence biological changes occur
-> The aim of the PTH is to increase the serum Calcium level

Calcitonin :-

-> It is produce from the parafollicular cells of thyroid gland
-> Its action is antagonistic to parathyroid hormone
-> It causes calcification of bone by increasing the activity of osteoblasts
-> It decreases bone resorption and increases calcium excretion in Urine
-> It has decreased effect on serum calcium level

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SHORTS :-

3h) carbohydrates are classified into Monosaccharides, Disaccharides and polysaccharides.

→ It is divided based on number of types of carbohydrates present

Polysaccharides → Homoglycans
→ Heteroglycans

Homoglycans :- contains the same type of carbohydrates.
eg: starch, Glycogen, inulin, chitin

Heteroglycans :- Agar, agarose gel, Mucopolysaccharides Glycoproteins and Mucoproteins

Mucopolysaccharides :-

→ Mucopolysaccharides are the hetglycoaminoglycans. or heteropolysaccharides
→ It contains both uronic acid and amino acid
→ Acetylated amino group, carbonyl group and sulphate groups are present
→ Due to the presence of charged molecules, these attract water molecules and produces and viscous fluid
→ Mucopolysaccharides when combines with proteins forms mucoproteins.
→ examples of mucopolysaccharides : Heparin, Hyaluronic acid, derman sulphate, keratin sulphate

Heparin :-

→ It is a blood coagulant used in vitro condition to extract blood out for clinical studies
→ It is also used in vivo under suspected thrombo embolic condition to prevent intravascular coagu cation.

Hyaluronic acid :-

→ It is present in synovial fluid, blue bones, vitreous humours, it contains N-acetyl glycosamin, Beta glucoronic acid, Beta 1,3 N-acetyl glucosamine

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units.

Keratin sulphate:-

It is the only GAG which do not contain uronic acid

It is present in cornea and tendons

It contains galactose and N-acetyl-glucosamine units.

Phospholipids:-

→ It contains glycerol, ~~fatty~~ amino acids and Nitrogen bases

Phosphatides:-

→ phosphatides are the simples phospholipids.

→ They are derived from phosphatides.

→ They have one-carbon assymetrical and shows D and L forms.

Amphipathic:-

→ They are amphipathetic

→ They contain both hydrophobic and hydrophilic components.

Micelle formation:-

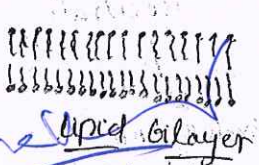
→ hydrophobic part is present away from the water and forms micelles

→ micelles help in better absorption and digestion of lipids.

Liposomes:-

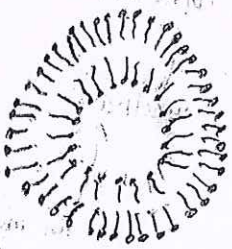
→ The lipid bilayer when it curls on itself then it forms liposomes/liposomes.

→ When they are present around enzymes or hormones they help in their transport towards the effector organs.



Lipid bilayer

micelle formation.



Liposome formation

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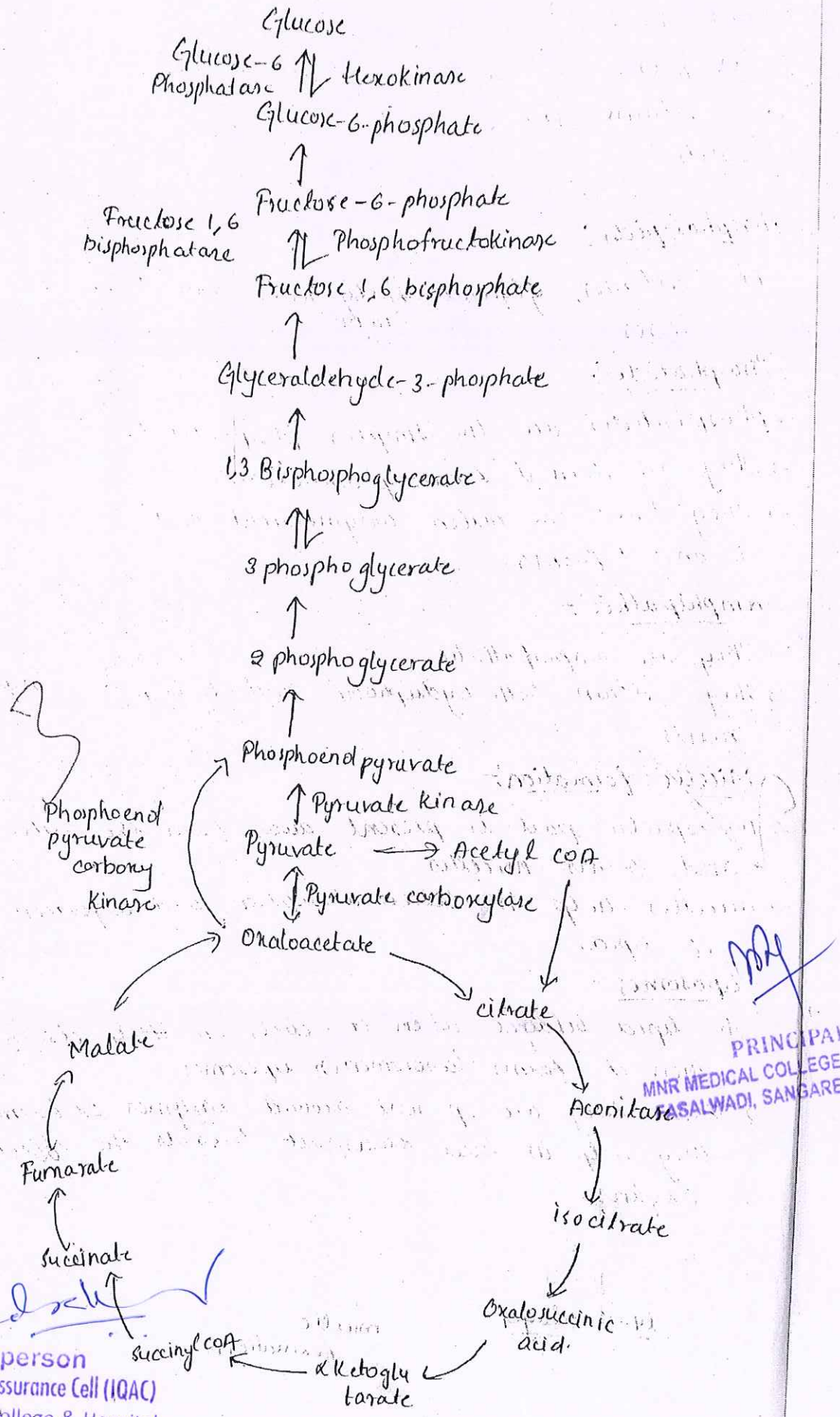
6) gluconeogenesis:- It is not the exact reverse reversal of glycolysis.



→ Carboxylation :-

Pyruvate carboxylates to form oxaloacetate catalysed by pyruvate carboxykinase

→ Malate aspartate shuttle :-

→ Through Malate aspartate shuttle oxaloacetate enters cytosol from mitochondria

→ Production of Fructose 1,6 bisphate :-

Upto fructose 1,6 bis phosphate reversal of glycolysis occurs.

→ Production of fructose 6 phosphate

Fructose 1,6 bisphosphatase catalyzes the conversion of fructose 6 phosphate to fructose 1,6 bisphosphate

→ Production of Glucose :-

→ from Glucose 6 phosphate, Glucose is produced, catalyzed by Glucose-6-phosphatase

---

Brief notes :-

13) Glucose → Pyruvate → Acetyl CoA → fat.

from fatty acid synthesis

~~Acetyl CoA~~ ←

Acetyl CoA $\xrightarrow[\text{transacetylase}]{\text{acetyl}}$ Acetyl-CE + CoA

Malony CoA + ACP-SH $\xrightarrow[\text{transacetylase}]{\text{Malonyl}}$ Malonyl-S-ACP + CoA

Acetyl CE + Malonyl ACP

↓ β ketoacyl synthase

Acetoacetyl ACP / β ketoacyl ACP.

↓ β ketoacyl reductase

Beta hydroxy. Octamethyl heptanoyl acp

↓ Enoyl synthase

Enoyl ACP

↓

Butyryl ACP. → Palmitic acid.

16) Galactosemia:-

→ It is due to the accumulation of Galactose-6-phosphate
→ It results in Hyperglycemia
→ Bilirubin uptake is reduced, Bilirubin conjugation is reduced, which increases the unconjugated Bilirubin in the body
→ causes liver inflammation, free Galactose accumulation causes Galactosemia
→ Galactose ⟶ dulcitol, dulcitol accumulation in the lens of the eye causes congenital cataract due to its osmotic effect
→ accumulation in renal tissue causes acidosis

Treatment:- Intake of lactose free diet, in the absence of treatment causes mental retardation.

---

17) Metabolic adaptation to starvation:-

→ During starvation there is decreased glucose levels in the body
→ at first it is compensated by glycogenolysis, this can compensate upto 18 hours of fasting
→ The primary requirement is to meet the needs of brain

Gluconeogenesis:-

→ even before glycogenolysis terminates gluconeogenesis accelerates
→ The substrates for gluconeogenesis is provided by amino acids produced by the muscles.
→ The amino nitrogen is transfered from other amino acids to pyruvate for alanine formation
→ alanine enters the liver and forms pyruvate for glucose formation.

Lipolysis:-

→ The high-increase in glucagon-insulin ration shunts down the glucose utilization of heart, kidney and skeletal muscle
→ fatty acids start mobilizing from the adipose tissue

→ By the action of glucagon fatty acids mobilize from adipose tissue
→ heart muscles, kidneys, brain and skelal muscles now depend on fatty acid for energy
→ carnitine acyl transferase regulates the beta oxidation
→ Increase in glucagon starts producing ketone bodies as energy fuel
→ Excess production of ketone bodies cause Metabolic acidosis
→ Metabolic acidosis and dehydration if not corrected properly leads to death

---

18) Gauchers diseases :-

→ In gba gauchers diseases, the enzyme affected is Beta glucosidase
→ Due to the impairment of Beta glucosidase, glucose-cerebroside will be accumulated
→ glucose-cerebroside is a lipid.
→ The symptoms of gauchers diseases / salient features :-
* Gauchers diseases is of 3 types :- infantile, adult, children. causing hepatomegaly, bone erosion and anaemia

---

19) Electrophoresis :-

→ Electrophoresis is a common analytical procedure which is used to seperate divide plasma proteins. / differentiate the plasma proteins.
→ Agar / paper electrophoresis with vellor bubber divides / seperates the plasma proteins into 5 distinct bands
→ The 5 distinct bands are albumin, α_1, α_2, β, γ globulin.
→ The concentration of this band fraction can be determined by using densitometer
→ (Electro) Electrophoresis is used to determine / diagnose many syndromes like
* Nephrotic syndrome
* α_1 antitrypsin syndrome
* Acute inflammations.

→ Metabolic acidosis

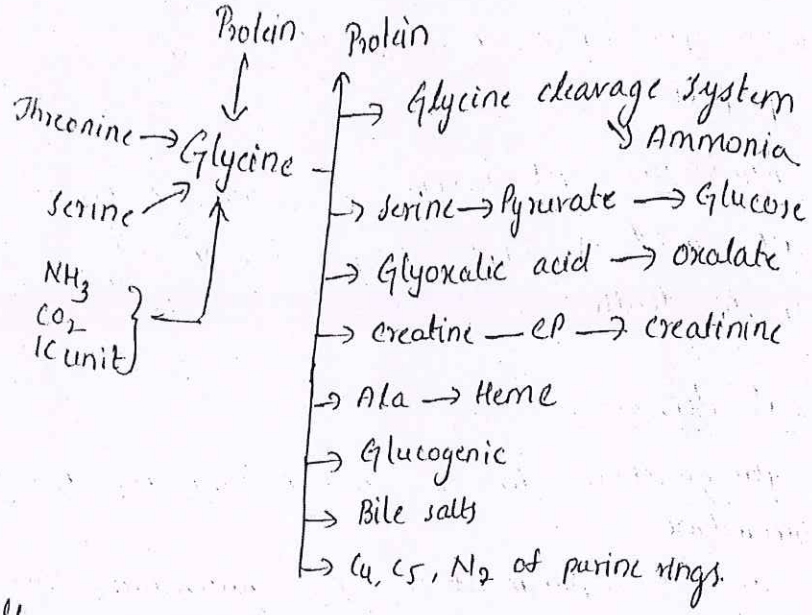
b) Ga

→ 2

→ 3

→ 5

20) Glycine :-



17) Metabolically Glycine produces :-

→ → Creatine, creatine phosphate, Creatinine

→ → Heme

→ a → Purine ~~rings~~ nucleotides.

→ Inhibor neurotransmitter

→ → Detoxyfication of benzoate

→ conjugated salts.

→ Glucogenic

→ → contribution to 1 carbon pool and Glycine cleavage system.

→ → conversion to serine

→

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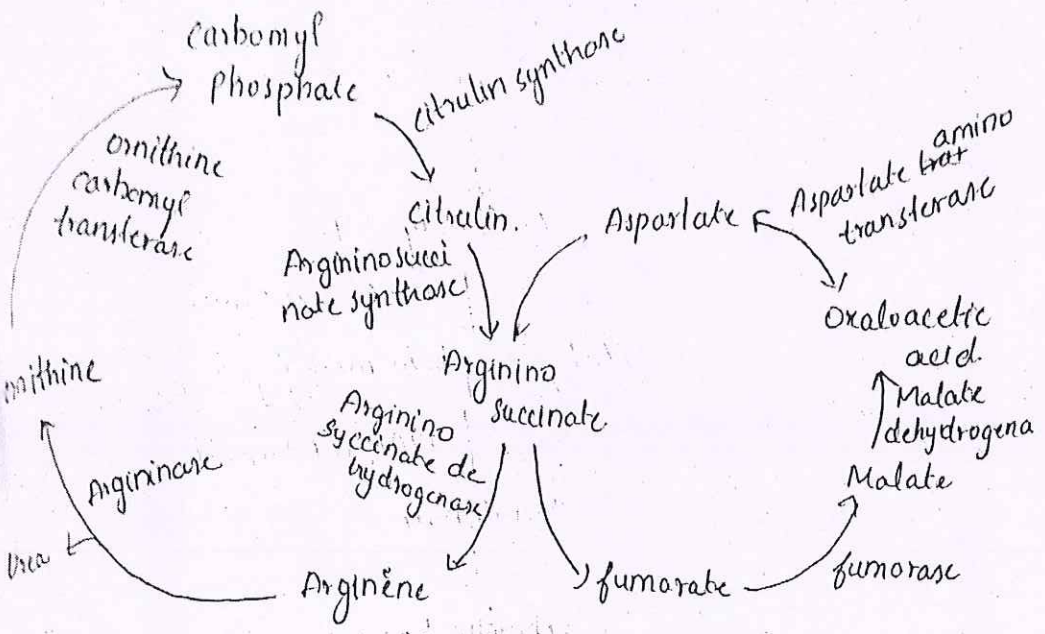
Note:-

a cycle is defective here



→ Ammonia is excreted as Urea from the body

→ If the above cycle is in defect then Urea will not produce from the arginine

→ Then ammonia will not be excreted from the body

→ Ammonia mainly affects the brain as it is accumulated there when the Urea cycle is not functioning properly

→ This cause elevated NH_3 levels in the body as carbomyl phosphate is formed from NH_3 and CO_2

$$NH_3 + CO_2 \xrightarrow{\text{Carbomyl phosphate synthase}} \text{Carbomyl phosphate}$$

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15) Bile salts and Bile pigments :-

→ Bile salts and pigments form from the exocrine part of the Pancreas

→ These are required for the digestion of carbohydrates, lipids, steroids and fats.

→ Cholesterol which produce from the dietary intake and intra hepatic tissue from the liver is either excreted as VLDL or enters the Bile acids / salts as.

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free fatty acids

→ Or cholesterol is converted into Bile salts / pigments.

---

14) Regulation of cholesterol :-

→

Acetyl CoA + Acetyl CoA

↓

Acetoacetyl CoA

↓ HMG CoA synthase

Betahydroxy Beta methylglutaryl CoA

↓ HMG CoA reductase

Mevalonate

↓

3 phospho 5 pyrophospho mevalonate

↓

Isopentenyl pyrophosphate

↓

Farnesyl pyrophosphate

↓

Squalene

↓

Lanosterol

↓

Desmosterol

↓

cholesterol

→ Upto HMG all the steps are similar to fatty acid synthesis and cholesterol synthesis

→ HMG CoA to Mevalonate is unique to cholesterol synthesis, so it is a rate limiting step in cholesterol

HMG CoA ⟶ Mevalonate is the rate limiting step in the cholesterol synthesis.

RE-TEST AND ANSWER SHEETS

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4. Evaluation of Corneal ulcer:

Clinical Evaluation:

1) Thorough history taking – to elicit mode of onset

2) General physical examination

3) Ocular examination –

a) Diffuse light examination – for lesions on lids, Cornea

b) Regurgitation test – to rule out lacrimal duct infection

c) Biomicroscopic examination – by staining ulcer.

Laboratory Investigations:

1) Routine lab investigations – TLC, DLC, ESR, Blood sugar

2) Microbiological investigations – to identify causative organisms

- Gram & Giemsa stain – for infecting organisms.
- Calcofluor white stain – for fungal Hyphae
- Culture on blood sugar – for aerobic organisms
- Culture on SDA medium – for fungi

Treatment – Uncomplicated Corneal Ulcer:

1) Specific treatment:

a) Topical antibiotics:-

- at early stages drugs for both gram (-ve) & (+ve) organisms.

- Fortified Cefazolin
- Fortified tobramycin } any ②
- Fortified vancomycin } every 1 minutes for 3 months.

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Subsequent therapy

- if no response to antibiotics, treatment according to culture

b) Systemic antibiotics:

- usually not required
- Cephalosporins & aminoglycosides - when scleral involvement & perforation is present

2) Non Specific treatment:

1) 1% atropine → to reduce pain & to increase blood supply
2) Systemic analgesics - paracetmol & ibuprofen & anti inflammatory drugs
3) Vit-A, B Complex - helps in healing

3) physical & general measures:

1) Hot fomentation
2) Dark goggles
3) Rest
4) good diet.

5. Iridocyclitis:

→ Inflammation of iris & pars plicata part of ciliary body are equally involved

Etiology:

- Idiopathic - 50%.

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- HLA - B27 - ankylosing spondylitis - young males
  - Inflammatory Bowel disease
  - Psoriatic arthritis
  - Reiter's syndrome
- Juvenile arthritis.

Symptoms:

i) Acute → photophobia
Red eye
lacrimation
Decreased vision

ii) Chronic → minimal symptoms
white eye

Signs:

1. Lid oedema
2. Circum Corneal Congestion
3. Cornea → Corneal oedema
   - Keratic precipitates - Diffuse
     - mutton fat
     - Small & medium
     - Old keratic precipitates

Anterior Chamber signs - aqueous cells
aqueous flare
Hypopion
Changes in angle of AC.

Iris signs - Loss of normal pattern
- Change in colour - muddy color

- Iris nodules - Koeppe's nodules
- Bu casaac's nodules.

Pupillary signs - Festoned pupil
- narrow pupil - oedema
- Sluggish pupillary reactions.

lens → Complicated Cataract

Treatment:

1) Nonspecific - 1% atropine - Spasm → reduce pain
→ Dilates & breaks synechiae
- reduce vascularity

- Corticosteroids - Betamethasone - to reduce glaucoma

Systemic → Corticosteroids - antifibrotic effect

2) NSAIDs - when associated with diseases
- If Rheumatoid arthritis - phenylbutazone
- If ankylosing spondylitis → Naproxen

- Immunosuppressive agents - Cyclosporin
- azythromycin

***physical measures:**

Hot fomentation - soothing effect
Dark goggles.

Specific treatment: for cause

Treatment for Complications:

1) Inflammatory glaucoma - timolol maleate
2) Cataract - lens extraction

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Ref.No.MNRMC/2022/020

Date:01/02/2022

CIRCULAR

The following is the repeat internal assessment examination schedule for the students who have not attended to even one internal assessment examination during the course of time. The students are 2020-21 batch are informed to approach the heads of the departments to appear.

Date	Department	Time
02/02/2022	Anatomy	09:00 am to 01:00 pm
03/02/2022	Physiology	09:00 am to 01:00 pm
04/02/2022	Biochemistry	09:00 am to 01:00 pm

Principal

Copy to:

- Medical Superintendent / CEO
- Assistant Director
- Chief Academic Officer
- Concerned Faculty
- HOD's of Concerned Dept.
- Chief Security Office
- Notice Boards

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